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Root defense responses to fungal pathogens: A molecular perspective

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Abstract

This review will focus on the molecular and genetic mechanisms underlying defense responses of roots to fungal pathogens. Soil-borne pathogens, including *Phytophthora, Pythium, Fusarium,* and *Bipolaris,* represent major sources of biotic stress in the rhizosphere and roots of plants. Molecular recognition and signaling leading to effective resistance has been demonstrated to occur between host and *Phytophthora,* or *Pythium*. The hypersensitive response and apoptotic cell death, two oxidative processes that limit biotrophic pathogens, generally act to exacerbate disease symptoms induced by necrotrophic organisms. Although pathogenesis-related proteins can be expressed in roots during pathogen challenge, salicylic acid has not been implicated in root-mediated interactions. Jasmonic acid and ethylene have been found to mediate parallel as well as synergistic pathways that confer partial tolerance to necrotrophic pathogens, as well as induced systemic resistance to root and foliar pathogens. Genomics approaches are revealing new networks of defense-signaling pathways, and have the potential of elucidating those pathways that are important in root-defense responses.

Abbreviations: CEVI – constitutive expression of VSPI (vegetative storage protein 1); COI1 – coronatine-insensitive 1; E – ethylene; EDS – enhanced disease susceptibility; EIN – ethylene-insensitive; ERF1 – ethylene response factor 1; ETR – ethylene resistant; *fad* – fatty acid desaturation; *Ggt* – *Gaeumannomyces graminis* var. *tritici*; HR – hypersensitive response; LRR – leucine-rich repeat protein motif; JA – jasmonic acid/jasmonate; NBS – nucleotide binding site protein motif; PR – pathogenesis-related; ROS – reactive oxygen species; SA – salicylic acid/salicylate; TLP thaumatin-like protein; PR-5

Introduction to root pathogens and the infection process

Like the above-ground organs, roots can be attacked by a number of pathogenic and parasitic organisms. These include, in order of importance, fungi, nematodes, bacteria, viruses, and parasitic higher plants. Monetary losses due to soil-borne pathogens of vegetables, fruits, and field crops have been estimated at US$4 billion annually in the US (Lumsden et al., 1995). Compared to infection by foliar pathogens, there are many important differences in the ecology, epidemiology, life cycles, pathogenesis, and infection caused by root pathogens. Within the last few years, there have been major advances in the understanding of host–pathogen interactions, mostly involving foliar pathogens. Less well understood are the interactions and mechanisms of resistance to necrotrophic root pathogens; these do not have the high degree of host specificity that characterize

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most biotrophic foliar and root pathogens. *Arabidiopsis thaliana* has become a model host plant, but few root pathogens have been used in this system.

The most significant pathogens of the roots of crop plants are either fungi or filamentous bacteria of the genus *Streptomyces* (Loria et al., 2003). There are a few pathogenic soil-borne bacteria, such as *Ralstonia solanacerarum,* which causes a wilt, and the well-studied *Agrobacterium tumefaciens,* which causes crown gall by genetic transformation of the plant (Gelvin, 2000; Schell, 2000). However, pathogenic bacteria (with the exception of *Bacillus),* are short-lived in the soil, and susceptible to desiccation and to the high degree of rhizosphere competition (Loria et al., 2003). The only viruses known to infect roots, such as nepoviruses and tobraviruses; are introduced by nematodes, or by zoosporic fungi such *Olpidium* which transmits lettuce big vein (Lot et al., 2002) and *Polymyxa,* the vectors of soil-borne wheat mosaic virus and barley yellow mosaic virus (Driskel et al., 2002).

This review will focus on root responses to soil-borne pathogenic fungi, which make up the bulk of microbes attacking the roots. Most root pathogens are necrotrophic, that is, they kill host tissue with toxins, peptide elicitors, or enzymes that trigger host cell lysis and death, thereby providing conditions favorable to pathogen growth. Classic examples are the Oomycete *Pythium* and the Basidiomycete *Rhizoctonia*. Although some species of these genera can infect above-ground parts under wet, humid conditions, they primarily attack roots and emerging shoots. Both genera show a preference for young, juvenile tissue as compared to older woody tissues with secondary wall thickenings, and both can attack germinating seeds in the soil, causing pre-or post-emergence damping-off or seedling rot. They also can attack young root tips and feeder roots, since the newest tissue is formed at the root tip. These pathogens can directly penetrate the root epidermis, and thick-walled resistant survival structures have evolved that are capable of surviving environmental extremes in a dormant state in the absence of a susceptible host. Of the fungi that parasitize root systems, surprisingly few are biotrophic, that is, require a living plant to parasitize and obtain nutrients. Some, such as *Phytophthora sojae,* are hemibiotrophic and form haustoria or feeding structures in plant cells. Another characteristic of most root necrotrophic pathogens is their wide host range. For example, *Pythium ultimum* has been recorded on over 100 genera of plants in the US (Farr et al., 1987). In contrast to biotrophic pathogens, the majority of root necrotrophic pathogens do not appear to have closely coevolved with a specific host, or to be distinguished by races that are virulent on specific genotypes, varieties, or cultivars of domestic plants, and avirulent on closely-related genotypes.

The following is a generalized life cycle of necrotrophic root rotting soil-borne pathogenic fungi. Such fungi can survive in the soil in a dormant, quiescent state, when environmental conditions are not suitable for growth, or when the host is not present. They must also withstand microbial degradation and lysis, parasitism and predation, constituting an important trophic level in the soil ecosystem. Therefore, in many fungi, a thick-walled resistant spore or structure has evolved to serve this survival function. These survival structures are often dark-colored or melanized, making them more resistant to microbial degradation. In the case of *Pythium,* sexual spores called oospores, or thick-walled sporangia serve this purpose (Hendrix and Campbell, 1973). *Rhizoctonia* survives as microsclerotia, structures composed of fungal plectenchyma, aggregated thick-walled cells, or as dark, swollen monilioid hyphae (Parmeter, 1970). Both structures are formed in rotting root tissue, which can also offer a degree of protection.

Once environmental conditions become favorable and a root emerges or grows in close proximity to the fungal propagule, the resistant structure will germinate to form hyphae that will grow toward the susceptible root or seed. If conditions are wet enough, *Pythium* will form zoospores, motile flagellated spores that swim in the film of water around soil particles, and contact the root. Fungi have mechanisms of chemotaxis and chemotropism, and sense root exudates such as sugars, amino acids, organic acids and fatty acids (Deacon and Donaldson, 1993; Donaldson and Deacon, 1993; Ruttledge and Nelson, 1997; Tyler, 2002). They can move or grow in response to gradients of these compounds. Electrostatic charge may also be an important sensory stimulus for swimming zoospores (Van West et al., 2002). Zoospores are often attracted to the zone behind

the root tip, root hairs, or the area where a secondary root emerges from the pericycle. Once the fungal hyphae or zoospore contacts the surface of the root, there probably is a recognition event on the part of both the fungus and plant. This also involves attachment of the fungus to the root. The zoospore will encyst, form a cell wall, and germinate to form an infection hypha. When a hypha contacts the root, it can form an appressorium, a swollen structure that attaches to the root and forms the infection hypha for penetration. *Pythium* and *Phytophthora* form these structures; *Rhizoctonia* forms multicellular infection cushions, which serve a similar purpose. In order to penetrate the host cell wall, fungal hyphae excrete cell-wall degrading enzymes such as pectinases and other pectic enzymes; hemicellulases, cellulases, and proteinases (Campion et al., 1997). These macerating enzymes result in cell death. Non-host-specific toxins may also be formed, although not much is understood about their existence or structure in root pathogens (Desilets et al., 1994). Once the pathogen gains ingress, it grows intracellularly in the cortex of the root, killing the tissue ahead of the advancing hyphae, and colonizing the root. New infections can be initiated on adjacent roots by hyphae or zoospores produced on the killed tissue.

Diseases caused by soil-borne pathogens are considered to be monocyclic. New roots on the plant can become infected from initial primary infection, but there is not much plant-to-plant spread in a single season, because of the limited distance that the inoculum travels in the soil. This is unlike polycyclic foliar diseases, which produce tremendous amounts of spore inoculum which spread from plant to plant by wind or rain in an exponential fashion in a single season. Finally, when the fungal mycelial biomass is increased and the root is killed, the fungus will produce survival structures in the root, such as oospores or microsclerotia. Other secondary pathogens and saprophytes will colonize the root, and *Pythium* and *Rhizoctonia* do not have a high level of competitive saprophytic ability against these other organisms. Thus, the strategy of the necrotrophic pathogen is to grow quickly and colonize the root ahead of secondary invaders, and then convert to survival structures.

One subset of necrotrophic pathogens, the wilt pathogens, has a more specialized life cycle with an adaptation to growth in the vascular system. These include the forma speciales of *Fusarium oxysporum* and species of *Verticillium*. The forma speciales have a limited and specific host range, and often form races. Wilt pathogens also colonize the cortex of the root, but gain access to the xylem in the zone of elongation before the vascular system is fully developed and differentiated, because the Casparian strip, suberized tissue in the endodermis surrounding the stele, presents a barrier to direct penetration of the vascular system in older parts of the roots. These fungal pathogens block the movement of water in the xylem by producing mycelia, spores, and high-molecular-weight polysaccharides in the xylem vessels, while degrading plant cell walls and releasing pectic substances and other polymers that can clog the vascular system and reduce its water-transport efficiency to the leaves.

Rhizosphere pathogens can induce defense responses in roots

Plants mount resistance to pathogens using a variety of mechanisms that can target specific or multiple pathogens. These mechanisms include the production of antimicrobial metabolites, inactivation of pathogen-derived toxins and lytic enzymes, and triggering of host-defense responses by pathogen-or host-derived elicitors. Processes that serve to rapidly limit growth of the pathogen at the site of infection are essential to disease resistance, and involve the generation of reactive oxygen species (ROS) that induce localized tissue collapse and necrosis. Also essential is a systemic resistance, mediated by host-derived salicylic acid (SA) that provides protection in the non-inoculated portions of the plant. Elicitation of general resistance mechanisms by the plant growth regulators jasmonic acid (JA) and ethylene (E) also contributes to disease resistance. Here, JA and methyl jasmonate (MeJ), a naturally occurring derivative of JA, will sometimes be collectively referred to as jasmonate.

Only a small number of defense pathways and resistance mechanisms described for leaf-pathogen interactions have been reported so far in roots (Figure 1). In response to challenge by necrotrophic fungal pathogens, roots typically exhibit the JA- and E-dependent defenses (Devoto and

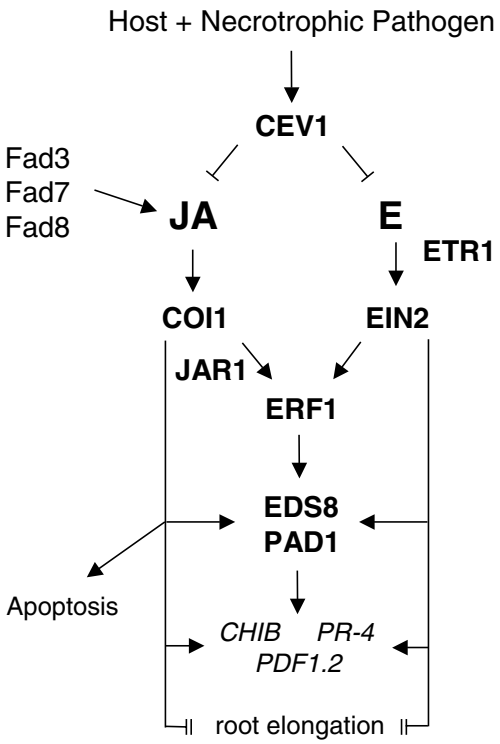

Figure 1. Proposed jasmonate (JA)-and ethylene (E)-mediated host responses to necrotrophic pathogens. Fatty acid desaturases (Fad3 Fad7 Fad8) and cellulose synthase (Cev1) are required for the wild type effects of JA and E on tolerance to such pathogens. Increased flux through either the JA or E pathway enhances pathogen tolerance, while modulating root growth and morphology.

Turner, 2003; Turner et al., 2002; Wang et al., 2002), but not genotype-specific resistance or the SA-dependent defense response. Although it is possible that roots lack effectors for triggering the SA pathway during invasion by necrotrophs, the absence of strong resistance might reflect the limited number of root–pathogen interactions that have been examined, and the difficulty in identifying genotype-specific interactions in below-ground pathosystems.

Interactions with Oomycete root pathogens are among the best studied with respect to elicitation of host defense responses by pathogen-derived factors. Many of these pathogens produce phytotoxins, enzymes that degrade host cell walls and phytoalexins, and inhibitors of pathogenesis-related proteins (Van West et al., 2003), reflecting the offensive–defensive nature of chemical signaling between plant and pathogen. *Pythium* and *Phytophthora* produce families of peptides or small proteins that can trigger a variety of host-defense responses. *Pythium oligandrum* produces an elicitor peptide, oligandrin, that stimulates host suppression of *Phytophthora parasitica* on stems of *Lycopersicon esculentum* (tomato) (Picard et al., 2000). Oligandrin induces the deposition of material at the host cell wall that blocks and contains the pathogen, and appears to disrupt hyphal wall synthesis. Picard and co-workers postulate that phytoalexins might be involved in the formation of host barriers. *Pythium aphanidermatum,* the causal agent of stalk rot and seedling damping-off in a broad range of hosts, secretes a protein that elicits programmed cell death and accumulation of the phytoalexin precursor 4-hydroxybenzoic acid (Viet et al., 2001) in the dicots *Nicotiana tabacum* (tobacco) and tomato, but not in the monocots *Zea mays* (maize), *Avena sativa* (oat), and *Tradescantia zebrina*. These findings suggest that host-dependent signal perception might determine whether the interaction will lead to susceptibility or resistance.

"Resistance" proteins associated with genotype-specific interactions, including proteins encoded by classical resistance (R) genes and the so-called pathogenesis-related (PR) proteins, are expressed in some root–pathogen interactions. The tomato *I-2* gene, conferring resistance to the wilt pathogen *Fusarium oxysporum* f.sp. *lycopersici,* encodes a member of the nucleotide-binding site/leucine-rich repeats (NBS-LRR) family of resistance proteins (Mes et al., 2000). *I-2* is expressed in lateral root primordia of young roots, and vascular regions of mature roots and foliar organs. The arrest of hyphal growth at the vascular interface is postulated to be the basis for *I-2*-mediated resistance against *F. oxysporum.*

Although the hypersensitive response (HR) has not been extensively examined in root–pathogen interactions, an interesting example of the HR in roots has been described for a soybean-*Phytophthora sojae* interaction (Kosslak et al., 1996). The investigators characterized a recessive mutation in soybean that resulted in the spontaneous formation of necrotic lesions on roots. Roots of NR (necrotic root) seedlings produced the expected HR in response to an incompatible strain of *P. sojae,* but sustained a four-fold reduction in disease incidence when inoculated with a compatible (disease-causing) strain.

Reduced incidence was temporally correlated with necrosis, and necrotic tissue was found to have high levels of two defense-related molecules, glyceollin and anionic peroxidase.

Despite the lack of strong evidence for an SA-dependent defense pathway in roots, accumulation of PR-l and PR-5, a thaumatin-like protein (TLP), has been reported in barley roots during challenge with *Bipolaris sorokiniana,* a necrotrophic root pathogen of *Triticum aestivum* (wheat) and barley (Liljeroth et al., 2001). These proteins accumulate in the root tips 6–10 days after inoculation with *B. sorokiniana,* and have also been detected in non-inoculated roots and leaves, indicating the movement of a signal that induces their expression in distal tissues. Similar patterns of induction have been observed in barley inoculated with *Blumeria graminis,* the causal agent of powdery mildew, and *Drechslera teres,* the net blotch pathogen, suggesting that the expression of PR-1 and TLP is part of a general defense response. In contrast to barley, induction of these proteins is not detected in wheat roots.

High levels of PR-l and TLP are correlated with high rates of cortical cell death (Liljeroth et al., 2001). Not surprisingly, infection by necrotrophs, including the take-all pathogen *Gaeumannomyces graminis* var. *tritici (Ggt),* is also correlated to higher rates of cortical cell death, possibly due to increased release of nutrients available to the pathogen, and a compromised ability of the host to mount a defense response. However, expression of PR-l and TLP in barley roots does not afford significant protection against necrotrophic pathogens.

General resistance mechanisms that simultaneously curtail biotrophic and necrotrophic pathogens (Heath, 2000; Thordal-Christensen, 2003) can be constitutive, thereby conferring an ongoing level of protection to the plant. For example, a PR-10-like protein is expressed in the root hairs and root epidermal cells of pea plants (Mylona et al., 1994), and is postulated to have a defense function in these tissues. Other PR and defense-related proteins have been detected in roots following pathogen infection, but do not confer significant levels of protection. Host-derived chitinase and glucanase accumulate around hyphae of *Fusarium culmorum* and *Fusarium avenaceum* that infect roots of susceptible spruce and pine (Asiegbu et al., 1999). Peroxidase activity also accumulates at root cell walls, presumably serving a role in cell-wall strengthening and free radical scavenging. The lack of effective resistance underscores the pathogen-specific nature of PR proteins, and the importance of timing of the expression of these proteins. Also, their induction might be occurring by default as a part of the wound or general stress responses, and they might have a protective role against microbes that would otherwise be pathogenic.

Jasmonate- and ethylene dependent signaling in root defenses

The action of JA confers a moderate degree of host tolerance to necrotrophic root pathogens, and plants that are compromised in JA biosynthesis or signaling show enhanced disease symptoms. The *Arabidopsis fad* triple mutant (*fad3-2 fad7-2 fad8*), deficient in biosynthesis of the JA precursor linolenic acid, is more susceptible to the root pathogen *Pythium mastophorum;* 90% of the *fad* plants showed disease symptoms, as compared to about 10% of wild-type plants (Vijayan et al., 1998). Roots of *fad* plants also harbor significantly more oospores of *Pythium*. Exogenously applied MeJ reduced the level of infection in *fad* roots, but did not affect the growth rate of the pathogen *in vitro*. Another class of *Arabidopsis* JA mutations, designated *jar1* (jasmonic-acid resistant), shows reduced sensitivity to jasmonate and deficient JA signaling (Staswick et al., 1998). Both the *fad* and *jar1* plants exhibit enhanced susceptibility to *Pythium irregulare*.

The *fad* triple mutant is unable to accumulate *LOX2* and *PDF1.2* mRNA following *Pythium* challenge (Vijayan et al., 1998), indicating that a functional JA pathway is required to promote the expression of these defense-related genes in roots. However, a causal link between accumulation of these transcripts and disease tolerance remains to be demonstrated.

A particularly revealing *Arabidopsis* mutant, *esa1,* displays enhanced sensitivity to necrotrophic but not biotrophic pathogens (Tierens et al., 2002). In *esa1,* induction of camalexin biosynthesis and expression of *PDF1.2* mRNA are delayed, providing indirect evidence for the involvement of JA in these responses. Functional ESA1 protein appears to be required for the action of ROS;

treatment with the ROS-generating herbicide paraquat activates *PDF1.2* mRNA accumulation in wild-type but not *esa1* plants (Tierens et al., 2002).

Using reverse genetics approaches similar to those described for the JA pathway, a number of laboratories have demonstrated that the gaseous plant growth regulator ethylene (E), mediates the localized HR response (necrosis), induction of PR proteins, general resistance to pathogenic fungi in susceptible or non-host interactions, and the wound response. The ethylene-response pathway has been reviewed in detail (Wang et al., 2002).

As for JA mutants, plants impaired in E perception show more severe disease symptoms in response to necrotrophic pathogens than do wild-type plants. Tobacco expressing a defective E signal perception component, Etr1-1, is rendered insensitive to ethylene (Knoester et al., 1998). The *etr1-1* plants show a higher incidence of stem necrosis and wilting when grown in non-pasteurized soil, and these symptoms are mimicked by inoculation of the roots with normally non-pathogenic *Pythium sylvaticum*. Symptomatic (wilted) stems of *etr1-1* tobacco harbor pathogenic strains of *Rhizopus stolonifer, Fusarium oxysporum,* and *Thielaviopsis basicola* (Geraats et al., 2002). The *etr1-1* plant is also more susceptible to necrotrophic fungi, including *B. cinerea, Cercospora nicotianae,* and the macerating bacterium *Erwinia carotovora,* but not the biotrophs *Oidium neolycopersici, Peronospora tabacina* and TMV (Geraats et al., 2003). Inoculation of *etr1-1 Arabidopsis* with the root pathogens *P. irregulare, P. jasmonium,* and *P. aphanidermatum* results in substantial wilting, necrosis and eventual plant death as compared to wild-type and mock-inoculated mutant plants (Geraats et al., 2002). *Ein2* mutants of *Arabidopsis,* defective in E signal transduction, also are more susceptible to *Pythium* (Geraats et al., 2002).

The effect of ethylene on defense responses and disease symptoms varies with the host plant. E-insensitive *Arabidopsis* and tobacco respond similarly to necrotrophic and biotrophic pathogens; however, *etr1-1 Arabidopsis* remains tolerant to one *F. oxysporum* isolate, whereas the tobacco mutant does not (Geraats et al., 2002), suggesting that the action of E can be influenced by genotype- or pathosystem-dependent factors. Soybean plants carrying mutations at two E-perception loci, Etr 1 and Etr2, display a more subtle loss of tolerance to *Rhizoctonia solani* (Hoffman et al., 1999). Furthermore, a susceptible genotype of *Arabidopsis* that carries *ein2-1* exhibits decreased symptoms when inoculated by a normally virulent strain of the soil-borne pathogen *Ralstonia solanacearum,* the causal agent of bacterial wilt (Hirsch et al., 2002).

JA and E have overlapping roles in general plant resistance, in some cases, providing a degree of pathogen-induced tolerance for otherwise susceptible hosts. It is not surprising that both of these phytohormones act synergistically in certain defense responses, such as the induction of mRNAs encoding PR-lb and thaumatin-like protein in tobacco roots (Xu et al., 1994), and ERFI (Lorenzo et al., 2003) in *Arabidopsis*. The molecular basis for the synergism is unknown, but might involve co-association of F-box proteins in the SCF-ubiquitin ligase complex, as proposed for JA and IAA (Gazzarrini and McCourt, 2003).

In addition to induction of defense genes, JA and E might be affecting defense-gene expression by modulating root development. Both metabolites inhibit root elongation, and in doing so, possibly divert metabolic resources from roots undergoing pathogen attack, while activating general resistance in those roots. The effect of E on root elongation is independent of COI1 (Ellis and Turner, 2002), suggesting that JA and E are acting on root morphology through independent, parallel pathways.

Systemic resistance induced by rhizobacteria

Interactions between roots and certain non-pathogenic soil-borne microbes, including members of the genera *Pseudomonas* and *Bacillus,* can trigger a systemic resistance to root and foliar pathogens in the host (Pieterse and van Loon, 1999). This process, known as induced systemic resistance (ISR), is distinct from foliage-mediated systemic acquired resistance by the absence of SA signaling in the root (Chen et al., 1999), the absence of local necrotic lesions on the leaves (Liu et al., 1995), and the inclusion of pathogens to which wild-type levels of tolerance in the plant are mediated by the JA and E pathways (Ton et al.,

2002b). Ecotypes of *Arabidopsis* that undergo systemic acquired resistance are not always able to exhibit ISR in response to root colonizing rhizobacteria (Ton et al., 1999), suggesting that these two types of induced resistance are not identical.

In solanaceous hosts, SA appears to have a role in leaf-localized ISR, depending upon the bacterial strain (Audenaert et al., 2002; Maurhofer et al., 1998), and ISR can be accompanied by induction of PR protein genes (Maurhofer et al., 1994; Park and Kloepper, 2000). In contrast, ISR in *Arabidopsis* is governed by the JA and E pathways, but not by SA. For instance, the protection conferred by *P. fluorescens* strain WCS417r to *P. syringae* pv. *tomato* is compromised in *jar1, etr1, ein, eds, eir1* (ethylene insensitivity in roots), and *npr1* Arabidopsis mutants (Iavicoli et al., 2003; Knoester et al., 1999; Pieterse et al., 1998; Ton et al., 2002a), indicating the requirement for JA- and E-signaling, and NPR1 in either the roots or shoots, or both. The requirement for NPR1 in ISR appears to be SA-independent; ISR is fully operable in a NahG host that has reduced levels of SA (Van Wees et al., 1997), and applied JA and 1-aminocyclopropane-l-carboxylate trigger ISR in NahG plants (Pieterse et al., 1998). Root-colonizing rhizobacteria that reduce symptoms of *P. syringae* pv. *tomato* in the *fad* triple mutant, *ein2,* and *npr1* (Ryu et al., 2003) suggest the existence of additional components or mechanisms for ISR that are distinct from the SA pathway.

Involvement of JA and E *per se* in the ISR has not been demonstrated. ISR in *eds4, eds8,* and *eds10* is not restored by applications of JA, although the E precursor l-aminocyclopropane-1-carboxylate restored ISR in *eds8* (Ton et al., 2002a), a JA pathway mutant. Neither JA nor E levels are significantly elevated in leaves of plants whose roots were treated with WCS417r, as compared to untreated plants. However, the conversion of l-aminocyclopropane-l-carboxylate to E by ACC oxidase is elevated in WCS417r-treated plants (Pieterse et al., 2000). The enhanced conversion is independent of ISR, as it occurs with variants of WCS417r that lack the ability to induce ISR, as well as in *jar1* and *npr1* hosts (Hase et al., 2003). Rhizobacteria-mediated ACC conversion is postulated to "prime" the host for stronger E-mediated defense responses upon pathogen challenge (Hase et al., 2003).

The ability to undergo ISR co-segregates with basal resistance to *P. syringae* pv. *tomato*. Classical genetics indicate that both traits are governed by a single dominant gene, called *ISR1* (Ton et al., 1999). Arabidopsis ecotypes that carry the recessive *isr1* allele exhibit JA-induced root stunting and *Atvsp* gene expression, suggesting that *ISR1* does not encode a component of the JA pathway (Ton et al., 2001). However, *isr1* plants display an attenuated E triple response and reduced expression of E-induced genes *hel* and *Pdf1.2*. Ton et al. (2001) hypothesize that the *isr1* genotype harbors reduced E sensitivity, and that ISR1 is involved in the E response pathway.

Although the molecular pathways and processes governing ISR in the leaf are being elucidated, those in the root remain obscure at present. Triggering of ISR in the root has been studied primarily in the context of biological control of root pathogens, and is proposed to involve siderophore-dependent ROS production in the host (Audenaert et al., 2002), and production of the antifungal metabolite 2,4-diacetylphloroglucinol by the biocontrol organism (Iavicoli et al., 2003). It is intriguing that both the biological control rhizobacterium and its target pathogen induce defense genes in cucumber roots (Chen et al., 2000), although this response in tomato roots is associated with specific bacterial strains (Audenaert et al., 2002). So far, studies on ISR have been limited to dicot hosts, and more information on root processes and genes that signal ISR in the below-ground portions of the plant needs to be pursued.

Future directions

Higher plants employ a number of molecular mechanisms for adaptation to abiotic and biotic stresses. As compared to most aerial portions of the plant, roots are morphologically simple organs that must nevertheless maintain nutrient and water utilization, anchorage, and other functions that support the entire plant, despite changes in soil temperature, salinity, water availability, and levels of toxic compounds. Leaves and stems exhibit diverse defense signaling pathways in response to necrotrophic pathogens;

however, root-defense responses appear to be mediated primarily by JA and E, which confer a general resistance that is relatively weak (Figure 1). The SA-mediated systemic acquired resistance that is commonly induced by foliar pathogens has not yet been demonstrated to occur in response to root pathogens and parasites, which would be expected if SA is exclusively synthesized in leaves and unidirectionally translocated through the phloem. *Arabidopsis* has been shown to display an effective foliar defense response to the biotroph *Peronospora parasitica* but in pathogen-challenged roots, the same plant fails to mount the cell death and ROS responses typical of the HR (Hermanns et al., 2003). On the other hand, roots are able to mount strong genotype-specific resistance against parasitic plants (Gowda et al., 1999) and nematodes, with evidence of an HR (Williamson, 1999), phytoalexin induction, and PR protein synthesis (Baldridge et al., 1998), and against hemibiotrophic soil-borne fungi such as *Phytophthora sojae* (Dorrance and Schmitthenner, 2000) and biotrophic fungi such as *Plasmodiophora brassicae* (James and Williams, 1980). It is possible that a limited set of genes and pathways are recruited in roots. This might be attributable to the buffering nature of soil (e.g., Loria et al., 2003), or reflect the need for a streamlined and rapid surveillance system that remains responsive to numerous stresses. Alternatively, organ specialization might necessitate limited expression of defense pathways in roots, resulting in repression of pathways that arose in the aerial organs after roots evolved from shoots and stems about 360 to 410 million years ago (Waisel et al., 2002).

It is of interest to determine whether molecular components of JA and E signaling identified in studies with foliar necrotrophic pathogens (Kunkel and Brooks, 2002) are also required for tolerance to root pathogens. Such components include ERF1 (Berrocal-Lobo et al., 2002), COI1 (Feys et al., 1994), an F-box motif-containing protein that is postulated to be a component of SCF-ubiquitin ligase complexes involved in protein degradation (Xie et al., 1998; Xu et al., 2002), and cellulose synthase CeSA3 (Ellis and Turner, 2001; Ellis et al., 2002). The causal relationship between cellulose synthesis and increased steady-state levels of JA and E remains to be determined. However, this finding, along with the enhanced transcription of JA-induced genes and atypical lignin deposition in the cell wall (Cao-Delgado et al., 2003) in *cev1,* suggests that flux through either JA or E or both are mediated by the cell wall or cell-wall-associated signals. Several other macromolecules associated with the cell wall, including cell wall proteins, transport complexes, and plasma membrane-localized kinase cascades (reviewed in Heath, 2000; Piffanelli et al., 1999), and actin (Sugimoto et al., 2000) are associated with biochemical processes involved in general resistance. These findings implicate the host cytoskeleton or a cytoskeletal element in defense responses.

Genomics approaches are helping to define whole pathways and cellular processes that underlie host resistance to foliar necrotrophic pathogens (Cheong et al., 2002; Ramonell et al., 2002; Sasaki et al., 2001; Schenk et al., 2000, 2003), and have the potential to do the same for root necrotrophic pathogens. In one comprehensive study, profiles of expressed genes were compared in 17 *Arabidopsis* mutants harboring various defects in the SA-, JA-, and E-mediated defense pathways (Glazebrook et al., 2003). By clustering differentially expressed genes according to their expression profiles, the researchers identified a group of JA-responsive genes that was only JA (COI1)-dependent, and a second group was jointly dependent upon E and SA via EIN2, EDS8, and PAD l. In this way, the expression of unique and common genes was correlated to the absence and presence of various components in the signaling pathways, and novel relationships between specific genes and signaling pathways components were identified.

Although some or many of the genes are likely to be significant in leaf–pathogen interactions, none were specifically earmarked as important for root defenses. A recent survey of wheat root ESTs expressed under several stress and non-stress conditions has revealed the presence of mRNAs involved in the HR and in the JA-, E-, and SA-signaling pathways (P. Okubara, unpublished data). However, the presence of mRNA in roots can sometimes be misleading, as in the case of those for PR-1b and thaumatin-like protein that are abundant, but not translated (Xu et al., 1994). Therefore accumulation and functional significance of these proteins in host defense need to be demonstrated.

The advent of molecular genetics and genomics is providing the means to identify genes in the pathogen that govern the infection process, pathogenicity strategies, and life cycle. Rapid and sensitive techniques such as real-time PCR will be useful for quantifying differences in the quality and timing of defense-gene expression that might indicate thresholds that delineate susceptibility and tolerance. With the exception of elicitors from *Pythium,* host recognition of necrotrophic pathogens has not been documented.

Despite extensive efforts by breeders, natural sources of resistance to many root pathogens remain elusive. So far, germplasm of the small-grain cereals, as well as close and distant relatives, does not confer adequate protection against most root necrotrophic pathogens. Presently, growers have limited options to control these diseases, as few fungicides are economical or effective against soil-borne pathogens.

Promising results in the area of enhanced resistance have recently been reported for foliar necrotrophic pathogens. Dickman et al. (2001) obtained tolerance to S. *sclerotiorum* and other pathogens of tomato by expressing genes that inhibit apoptotic host cell death. Although fungal growth was not directly inhibited, the pathogens failed to invade and kill the host tissue in the transgenic plants. Apoptosis, a mitochondrion-regulated process that occurs during normal root development (Gilchrist, 1998; Liljeroth and Bryngelsson, 2001), occurs during pathogen challenge (Lam et al., 2001), and in response to JA, E, and wounding. Apoptosis and the HR have been described in plants undergoing infection by root pathogens; *Sclerotinia sclerotiorum,* a broad host range necrotrophic root pathogen, induced nucleosomal laddering in infected tobacco leaves (Dickman et al., 2001). However, the role of this process in root defenses remains to be determined. By enhancing the flux through defense-signaling pathways or enhancing production of antimicrobial metabolites, such as saponins (Papadopoulou et al., 1999; Wilkes et al., 1999), and isothiocyanates (Tierens et al., 2001), a higher level of tolerance to necrotrophic root pathogens in adapted crop varieties might also be obtained.

The absence of recognition during the early stages of infection and/or the lack of an effective defense response suggests that plants have not yet developed or do not have resistance mechanisms against necrotrophs. Perhaps our understanding of older, more evolved pathosystems can provide leads to combat emerging diseases. Meanwhile, resistance has the potential to be a dominant component of the management of root pathogens, as it is with foliar pathogens.

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